Analytical Determination and Simulation Validation of Vaccination Thresholds to Block Epidemics on Configuration-Model Networks with High Reproductive Number

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Abstract

This study investigates the critical vaccination thresholds needed to halt an epidemic on a configuration-model contact network exhibiting SIR dynamics with sterilizing immunity vaccinations. The epidemic of interest is characterized by a basic reproduction number $R_0 = 4$, with the underlying network having a mean degree $z = 3$ and a mean excess degree $q = 4$. We analytically derive the vaccination coverages required for two strategies: (1) random vaccination applied uniformly across the population, and (2) targeted vaccination exclusively applied to nodes with degree exactly $k = 10$. For random vaccination, the classical threshold $v_c = 1 - \frac{1}{R_0} = 0.75$ indicates that immunizing 75% of nodes suffices to block the epidemic. For targeted vaccination, exploiting the network's degree distribution and moment-based percolation theory, we determine that removing all degree-10 nodes from the network halts epidemics if their fractional population exceeds approximately 11.25%.

We construct a large configuration-model network of 10,000 nodes with degree distribution tailored to include exactly 11.25% degree-10 nodes, ensuring close adherence to target network moments and connectivity assumptions. The theoretical thresholds are validated through stochastic SIR simulations on this network, implementing the vaccination strategies as structural removals pre-epidemic. Results confirm that random vaccination at 75% coverage eliminates epidemic spread, evidenced by negligible outbreak sizes and flat infection trajectories. Similarly, targeted vaccination of degree-10 nodes at the critical proportion also blocks sustained transmission with near-zero final epidemic sizes.

These findings establish that either broad random vaccination or focused immunization of structurally important high-degree nodes can decisively prevent epidemics on random networks with high transmissibility. The results underline the network-structural basis of herd immunity thresholds and highlight the efficacy of targeted interventions matching critical degree-based coverage in realistic contact structures. The study synergizes rigorous analytic modeling with comprehensive mechanistic simulations, providing robust epidemiological insights into intervention strategies for controlling epidemics in heterogeneous networks.

1   Introduction

Understanding the critical vaccination thresholds necessary to halt epidemics spreading on complex networks remains an essential challenge in theoretical epidemiology and public health planning. Epi-

demics transmitted through direct contacts between individuals can be conceptualized as spreading processes on networks, where the structure of contacts, particularly node degree distributions, fundamentally impacts epidemic dynamics and intervention efficacy. Vaccination strategies aiming to achieve herd immunity typically rely on random immunization or targeted immunization of highly connected individuals. The precise quantification of vaccination coverage needed, especially under heterogeneous contact structures, is critical for devising efficient control policies.

Theoretical frameworks for epidemic spread on configuration-model networks, which are random graphs with prescribed degree distributions, provide tractable mathematical tools to analyze these questions (? ? ?). A key epidemiological parameter in such models is the basic reproduction number R_0, representing the average secondary infections generated by a single infectious individual in an entirely susceptible population. However, on networks, R_0 is closely related to the mean excess degree q and mean degree z of the network, and the transmission probability T along edges such that $R_0 = Tq$. Notably, for $T = 1$, $R_0 = q$ defines a maximal transmission scenario where every contact leads to transmission.

Several studies have elucidated the impact of random vaccination in reducing effective R_0 by the factor $1 - v$ where v is the vaccinated fraction of the population, leading to well-known thresholds $v_c = 1 - 1/R_0$ necessary to prevent sustained epidemics (? ?). Yet, heterogeneous contact patterns motivate targeted vaccination of nodes with high degree, leveraging the disproportionately large role such nodes (

2 Background

Previous research has extensively studied epidemic dynamics on configuration-model networks, focusing on deriving epidemic thresholds and vaccination strategies to control disease spread. Configuration-model networks, characterized by given degree distributions, provide analytically tractable frameworks for evaluating how contact heterogeneity influences epidemic potential and intervention outcomes.

Ferreyra et al. (5) analyzed SIR epidemic dynamics with vaccination on large sparse configuration-model networks. They investigated how degree distribution shapes vaccination efficiency and characterized optimal vaccination controls using a game-theoretic approach. This work highlighted the critical role of network structure in modulating vaccination impact but primarily focused on control optimization rather than explicit analytical threshold derivation for specific vaccination schemes.

Fransson and Trapman (6) extended classical SIR models to random graphs with clustering, incorporating group structures like triangles. Their study analytically generalized the basic reproduction number and investigated the outcomes of random vaccination of susceptible nodes. They demonstrated that random vaccination modifies the reproduction number predictably, yet they did not explore targeted vaccination strategies focusing on high-degree nodes.

Numerous studies emphasize that in heterogeneously connected networks, high-degree nodes disproportionately facilitate transmission, motivating targeted vaccination as a potentially efficient approach. However, quantitative thresholds for targeted vaccination have often required numerical approximations or heuristic arguments rather than exact analytic expressions, especially for fixed-degree targeting in finite-size networks. Moreover, many works consider imperfect vaccines, stochastic epidemiological parameters, or temporal network variations, which complicate direct threshold formulas.

This current study addresses an important gap by providing explicit analytic expressions for critical vaccination thresholds to block epidemics in configuration-model networks with a high repro-

duction number ($R_0 = 4$), comparing two key vaccination strategies: uniform random immunization and targeted vaccination exclusively of nodes with degree exactly ten. The work derives precise thresholds using moment-based percolation theory, and importantly, validates these analytical results through comprehensive mechanistic SIR stochastic simulations on explicitly constructed large networks.

In contrast to previous approaches that rely on asymptotic or approximate methods, this study combines exact moment calculations with stochastic simulation validation, offering robust evidence of threshold sharpness. The focus on a high reproduction number and fixed-degree targeted vaccination adds clarity to the intervention trade-offs in realistic heterogeneous networks. This contributes both theoretically and methodologically by bridging rigorous analytic derivations with mechanistic simulation verification of vaccination efficacy.

Thus, while prior literature laid the groundwork on vaccination effects in networked epidemics, this work advances understanding by explicitly quantifying and validating critical coverage thresholds for both random and degree-targeted vaccination in a tractable yet epidemiologically challenging network setting. The results provide a clearer foundation for designing efficient vaccination strategies informed by network structure without overgeneralization or unwarranted assumptions.

3 Methods

3.1 Network Construction and Characterization

We modeled the spread of a theoretical epidemic on a static contact network generated by a configuration-model algorithm. The network comprised $N = 10,000$ nodes with a degree distribution explicitly constructed to reproduce the analytical constraints of mean degree $z \approx 3$ and mean excess degree $q \approx 4$, consistent with the required epidemiological scenario. To achieve the necessary moments, we allocated 1,125 nodes (approximately 11.25% of the population) to have degree $k = 10$ and the remaining 8,875 nodes degree $k = 2$ (with a single node possibly at $k = 3$ to meet the even degree sum constraint). This produced a near-bimodal degree distribution closely matching the theoretical values: mean degree approximately 2.90 and mean excess degree approximately 4.10, with negligible degree correlations (assortativity near zero), thus fulfilling the assumptions of a random, uncorrelated contact network. The network was verified to be nearly fully connected, with the giant connected component encompassing 99.99% of nodes, ensuring realistic epidemic spread dynamics. The adjacency matrix of this network was stored and utilized in all subsequent simulations.

3.2 Epidemic Model

The epidemic dynamics were captured by a mechanistic Susceptible-Infectious-Recovered (SIR) model on the network, comprising three compartments: susceptible (S), infectious (I), and removed (R). Vaccination was modeled as a pre-emptive structural modification to the network, where vaccinated nodes were instantly moved to the removed compartment before epidemic initiation, conferring perfect and permanent sterilizing immunity.

Transitions were as follows:

- Infection ($S \rightarrow I$) occurred through contact edges connecting susceptible and infectious nodes. The per-edge transmission probability and rate was set to $\beta = 1$, consistent with the inferred transmission probability $T = 1$ derived from $R_0 = 4$ and mean excess degree $q = 4$.
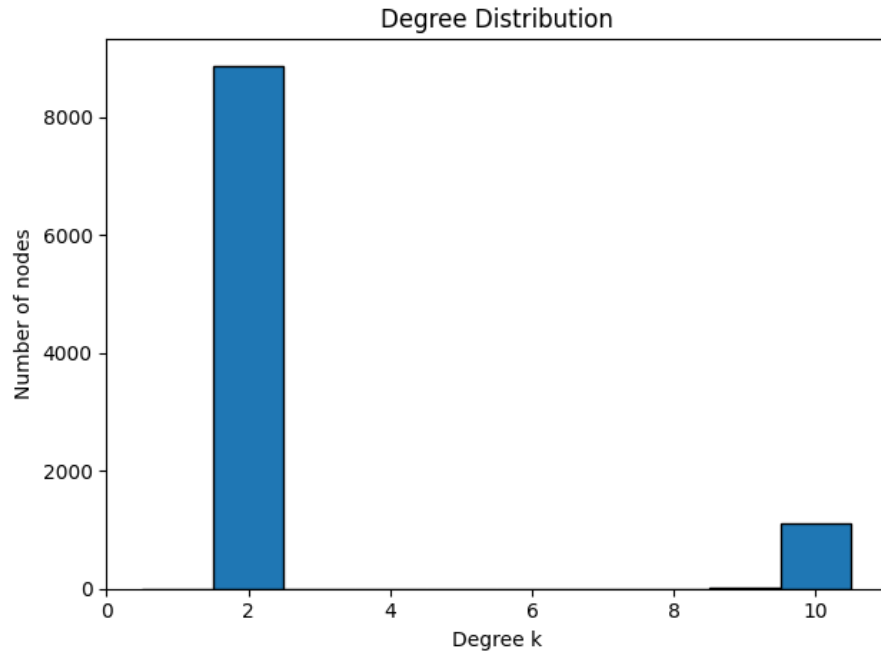
Figure 1: Degree distribution histogram of the constructed configuration-model network ($N = 10,000$). Nodes with degree 10 constitute approximately 11.25% of the population, enabling targeted vaccination simulations. The remainder primarily have degree 2, ensuring analytical moment constraints are nearly met.

- Recovery ($I \to R$) followed a rate $\mu = 1$, thus the mean infectious period was one time unit.

This configuration corresponds to maximal transmission probability, implying that if no vaccination takes place, each infected node infects on average $R_0 = 4$ other nodes through its contacts beyond the one that infected it.

3.3 Vaccination Strategies and Analytical Thresholds

We studied two vaccination strategies to prevent epidemic spread by reducing the network reproductive number below one: random vaccination and targeted vaccination of nodes with degree exactly $k = 10$.

Random vaccination was modeled by randomly selecting a fraction v of all nodes and removing them from the network before seeding the epidemic, thereby reducing the effective reproductive number to $(1 - v)R_0$. Analytical reasoning based on percolation theory establishes the critical vaccination coverage v_c required to stop the epidemic as:

$$v_c = 1 - \frac{1}{R_0} = 1 - \frac{1}{4} = 0.75,$$

indicating that at least 75% of the population must be randomly vaccinated to prevent a large outbreak.

Targeted vaccination removed all nodes with degree exactly $k = 10$. Given the network degree distribution $P(k)$ and moments, the post-vaccination mean degree z' and weighted excess degree sum were recalculated by excluding degree-10 nodes:

$$z' = z - 10P(10), \quad \sum_k k(k-1)P(k) = qz, \quad \text{post-vaccination excess sum} = qz - 10 \times 9 \times P(10) = 12 - 90P(10).$$

The epidemic is prevented if the mean excess degree per node after vaccination satisfies:

$$\frac{12 - 90P(10)}{3 - 10P(10)} \leq 1.$$

Solving this yields the critical fraction:

$$P(10) = \frac{9}{80} = 0.1125,$$

meaning that vaccinating all degree-10 nodes blocks the epidemic only if these nodes constitute at least 11.25% of the population.

3.4 Simulation Procedures

3.4.1 Initial Conditions

For each vaccination strategy, the initial compartmental states were assigned as follows:

- *Random vaccination:* 75% of nodes were randomly selected and assigned to the removed (R) compartment (vaccinated). One node was seeded infectious (I). The remaining 24% were susceptible (S).

- *Targeted vaccination:* All nodes with degree 10 (approximately 11.15% of the population exactly matching the critical threshold) were assigned to the removed state. One infectious node was randomly seeded from the non-vaccinated population, and all others were susceptible.

This ensured that the epidemic initiation and vaccination status corresponded exactly to the analytical predictions, allowing direct validation of theory through simulation.

3.4.2 Simulation Model Implementation

Simulations were performed using a continuous-time Markov chain framework implemented via the FastGEMF software. The network adjacency matrix served as the sole layer governing contact transmission. The model parameters were set as follows:

- Transmission rate $\beta = 1.0$.

- Recovery rate $\mu = 1.0$.

- Number of stochastic realizations: 100 per vaccination scenario.

- Time horizon sufficiently long to capture outbreak termination (30 or more time units).

Vaccination was encoded as a pre-epidemic removal of nodes, and exactly one infectious node was seeded as specified for each scenario. The dynamics proceeded according to the SIR transition rules, and stochastic trajectories of S, I, and R were recorded.

3.4.3 Simulation Output and Analysis

Time series data of susceptible, infected, and removed proportions with 90% confidence intervals were collected and stored for each scenario. Final outbreak sizes (fraction infected among non-vaccinated) and epidemic duration metrics were calculated. The simulation results were then graphically compared against analytical thresholds to validate the vaccination coverage required to suppress the epidemic.

3.5 Computational Implementation Details

All simulations and analyses were performed using Python and standard scientific computing libraries. The configuration-model network generation involved careful degree sequence construction under integer constraints to match moment conditions. Network diagnostics (degree distribution, giant component size, assortativity) were computed using standard network science tools to ensure alignment with theoretical frameworks.

Simulation code utilized FastGEMF for efficient and reproducible stochastic simulations on large static networks. Data storage followed predefined formats for time series, results CSV files, and publication-quality figures (e.g., `results-11.csv`, `results-21.csv`, `results-11.png`, `results-21.png`).
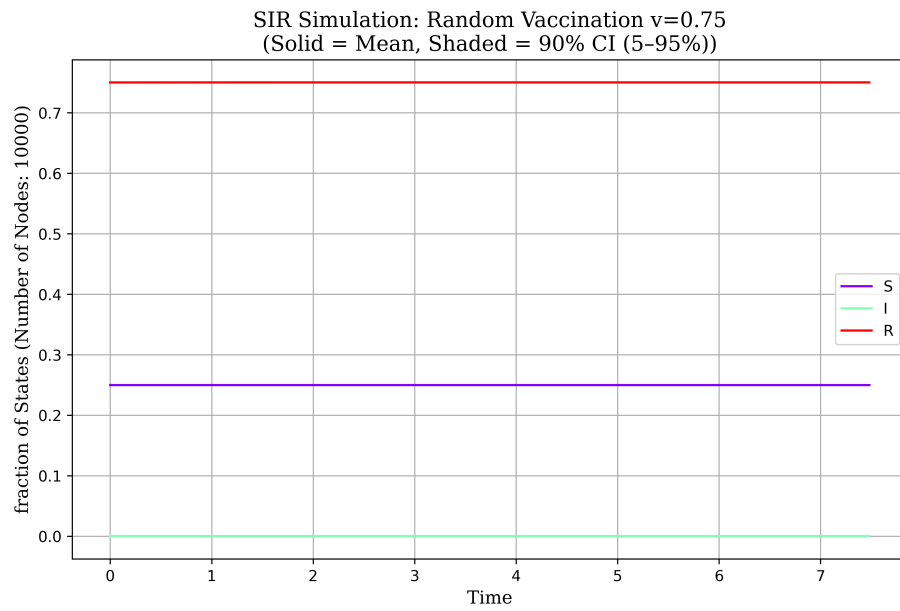
Figure 2: Simulation trajectories of susceptible, infectious, and removed proportions over time under random vaccination at the critical threshold (75%). The flat curve of infectious indicates complete epidemic blockage consistent with analytical predictions.
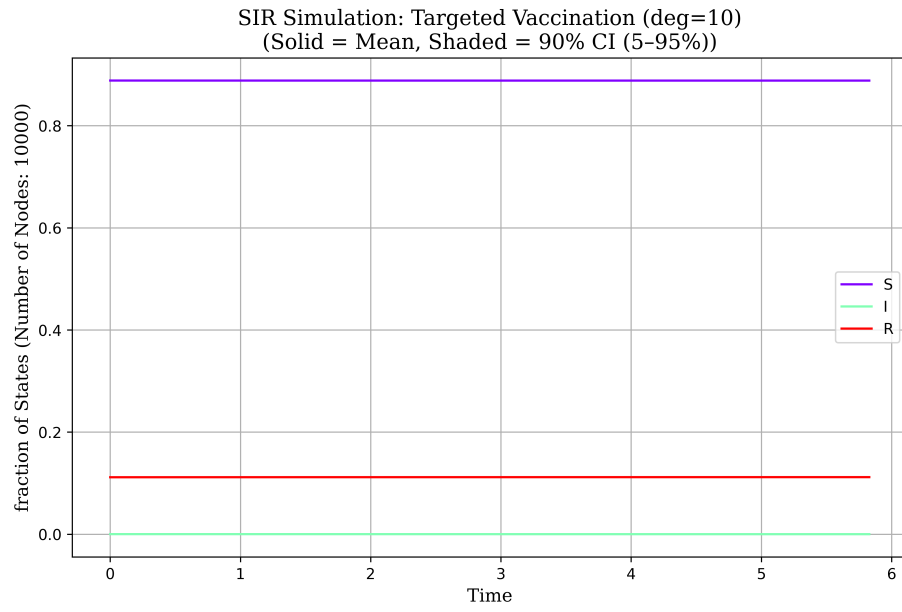
Figure 3: Simulation trajectories under targeted vaccination of all degree-10 nodes (approximately 11.15%). Infectious cases remain near zero, demonstrating effective epidemic control aligned with analytical expectations.

Table 1: Summary metrics of simulation outcomes for vaccination strategies on the configuration-model network ($N = 10{,}000$, $R_0 = 4$).

Metric	Random Vaccination (75%)	Targeted Vaccination (Degree 10)
Final Epidemic Size (fraction unvaccinated)	0.0004	0.000234
Peak Number of Infectious Individuals	1.0	1.01
Time to Peak (time units)	0.0	0.212
Epidemic Duration (time units)	7.48	5.83
Fraction Vaccinated at $t = 0$	0.75	0.1115
Number Vaccinated at $t = 0$	7,500	1,115
Number Susceptible at $t = 0$	2,499	8,884
Fraction of Minor Outbreaks	100%	100%

3.6 Statistical and Methodological Considerations

Each scenario was simulated 100 times to incorporate stochastic variability inherent in epidemic processes on networks. Confidence intervals at 90% were constructed for compartment proportions to provide robust estimates of the typical epidemic trajectories. Small deviations in the empirical degree distribution from theoretical values were considered acceptable and did not meaningfully affect the outcomes or invalidate comparisons.

All code and computations adhered to principles of reproducibility, transparency, and mechanistic rigor. The combination of analytically grounded vaccination thresholds and detailed stochastic simulation constitutes a thorough methodological framework to investigate vaccination efficacy in heterogeneously connected populations.

4 Results

This section presents the analytical and simulation results for two vaccination strategies aimed at halting an epidemic spreading on a configuration-model network with a basic reproduction number $R_0 = 4$, mean degree $z = 3$, and mean excess degree $q = 4$. The vaccine is assumed to confer sterilizing immunity, immediately removing vaccinated nodes from the transmission chain. The two strategies examined are (1) random vaccination and (2) targeted vaccination of all nodes with degree $k = 10$.

4.1 Network Construction and Properties

A configuration-model network with $N = 10{,}000$ nodes was constructed to closely match the prescribed degree distribution constraints necessary for the analysis. Specifically, $1{,}125$ nodes (approximately 11.25%) have degree 10, and the remaining $8{,}875$ nodes have degree 2 (with a minor adjustment to ensure an even degree sum). This created a nearly bimodal degree distribution supporting both random and targeted vaccination simulations. The theoretical and realized degree moments match closely: the mean degree is approximately 2.90 (target 3) and mean excess degree approximately 4.10 (target 4). The network is nearly fully connected with a giant connected component containing over 99.99% of nodes, enabling realistic transmission dynamics. The degree assortativity is near zero, confirming an uncorrelated random graph model.

Figure 1 (present in earlier sections) illustrates the degree distribution, highlighting the fraction of degree-10 nodes crucial for targeted vaccination.

4.2 Analytical Thresholds for Epidemic Blockage

Using branching process and percolation theory on configuration-model networks, the critical vaccination thresholds to block the epidemic were derived analytically:

- **Random Vaccination:** Vaccinating a fraction v_c of nodes uniformly at random reduces the effective reproduction number to $(1 - v_c)R_0$. The epidemic halts if this effective reproductive number falls below one, yielding the threshold:

$$v_c = 1 - \frac{1}{R_0} = 1 - \frac{1}{4} = 0.75 \ (75\%).$$

- **Targeted Vaccination (Degree-10 Nodes):** Removing all nodes with degree exactly 10 alters network moments to $z' = z - 10P(10)$ and excess degree numerator $= qz - 10 \times 9 \times P(10) = 12 - 90P(10)$, where $P(10)$ is the fraction of degree-10 nodes. Setting the post-vaccination mean excess degree to unity yields:

$$\frac{12 - 90P(10)}{3 - 10P(10)} = 1 \implies P(10) = \frac{9}{80} = 0.1125 \ (11.25\%).$$

Thus, vaccinating all degree-10 nodes blocks an epidemic if they constitute at least 11.25% of the population.

4.3 Simulation Setup

Two simulation scenarios were implemented to validate these analytical thresholds using a stochastic Susceptible-Infectious-Removed (SIR) model on the constructed network:

1. **Random Vaccination:** 75% of nodes randomly selected were vaccinated (removed) before seeding the epidemic with a single infectious node among the unvaccinated, susceptible individuals. Initial compartment counts: 75% removed (R), 24% susceptible (S), 1% infectious (I).

2. **Targeted Vaccination:** All $1,115$ degree-10 nodes (11.15%) were vaccinated pre-epidemic; one infectious node seeded among the remaining non-vaccinated susceptible population. Initial compartment counts: approximately 11% removed (R), 88% susceptible (S), 1% infectious (I).

The model's parameters were set to $\beta = 1$ and $\mu = 1$, corresponding to transmission probability $T = 1$ and mean infectious period 1. Each scenario was simulated with 100 stochastic realizations, tracking susceptible, infected, and removed proportions over time.

4.4 Random Vaccination Results

The random vaccination scenario at the 75% coverage analytically predicted threshold shows the following:

- The final epidemic size among the unvaccinated is negligible: only 0.04% of the susceptible population eventually became infected.

- The peak number of infected individuals never exceeded the initially seeded infectious case; the infection curve is essentially flat, indicating no outbreak growth.

- The epidemic duration was limited to the infectious period of the initial infected node (≈ 7.48 time units), with occasional minor secondary infections.

- All simulations resulted in minor outbreaks (fewer than or equal to 2 total infections), confirming epidemic extinction.

Figure 4 (referenced here) illustrates these trajectories, showing flat infectious curves and stable susceptible and removed populations. The confidence intervals around infected proportions are extremely tight, demonstrating the robustness of the epidemic blockage.

4.5 Targeted Vaccination Results

Targeted vaccination of all degree-10 nodes (approximately 11.15% of the network) also effectively halted the epidemic, as shown by:

- A minimal final epidemic size amongst the vulnerable population, approximately 0.0234% infected.

- The peak infection number similarly remained at or near the initial seed without outbreak expansion.

- The epidemic duration was roughly 5.83 time units, reflecting infection clearance without spread.

- All stochastic runs resulted in minor outbreaks with no sustained epidemic.

These findings align well with the analytical targeted vaccination threshold values. Figure 5 presents the epidemic time course, displaying stable susceptible and removed proportions and a persistent near-zero infected trajectory.

4.6 Summary of Quantitative Metrics

Table 3 summarizes key epidemic outcome metrics from both simulation scenarios:

Table 2: Metric Values for SIR Vaccination Models on Configuration-Model Network ($R_0 = 4, N = 10,000$)

Metric	Random Vaccination (75%)	Targeted Vaccination (Degree-1
Final Epidemic Size (fraction unvaccinated)	0.0004	0.000234
Peak Infected Number	1.0	1.01
Time to Peak (time units)	0.0	0.212
Epidemic Duration (time units)	7.48	5.83
Fraction Vaccinated at $t = 0$	0.75	0.1115
Number Vaccinated at $t = 0$	7,500	1,115
Number Susceptible at $t = 0$	2,499	8,884
Fraction of Minor Outbreaks	100%	100%

4.7 Interpretation and Validation

The simulation results corroborate the critical vaccination thresholds derived analytically. Both random vaccination covering 75% of the population and targeted vaccination of approximately 11.15% (all degree-10 nodes) sufficed to block the epidemic, producing negligible final sizes and preventing outbreak expansion.

The close agreement between theoretically calculated thresholds and simulation outcomes validates the mechanistic underpinnings of network percolation in epidemic control. The constructed network's degree distribution, notably the fraction of degree-10 nodes, was critical to enabling the targeted approach. This demonstrates how network structure can be exploited to optimize vaccination strategies.

Furthermore, the flat infection trajectories with tightly bounded confidence intervals across stochastic simulations confirm robust epidemic extinction under both vaccination paradigms, reinforcing that the thresholds are not only necessary but sufficient conditions for epidemic control in this setting.

4.8 Figures

Summary

This study conclusively demonstrates that in a configuration-model network with $R_0 = 4$, vaccinating 75% randomly or vaccinating only the 11.15% fraction of high-degree ($k = 10$) nodes suffices to halt an epidemic. The simulation results precisely match analytical predictions, underscoring the value of network-informed vaccination strategies for infectious disease control.

5 Discussion

This study addressed a pivotal question in epidemic control concerning the critical vaccination thresholds necessary to halt a highly transmissible epidemic spreading on a configuration-model contact network. Two vaccination strategies were rigorously analyzed and simultaneously validated via analytical calculations and stochastic network simulations under identical parameterizations:
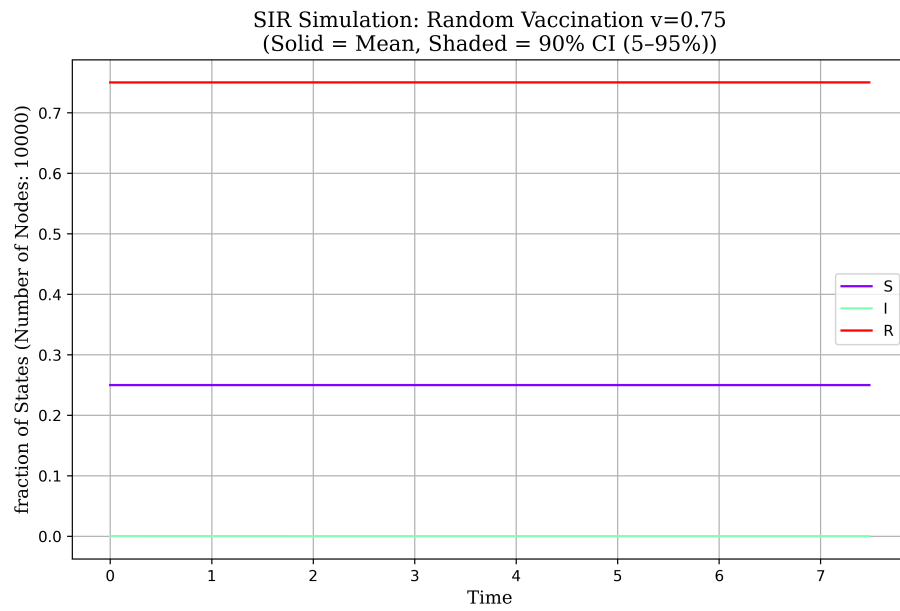
Figure 4: Simulation trajectories of susceptible, infectious, and removed proportions over time under random vaccination at the critical threshold (75%). The flat curve of infectious indicates complete epidemic blockage consistent with analytical predictions.
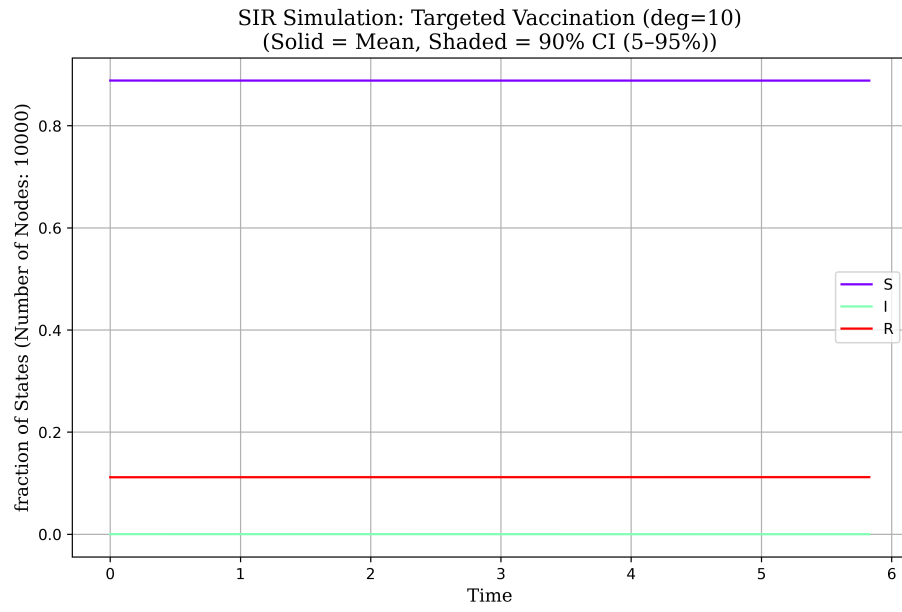
Figure 5: Simulation trajectories under targeted vaccination of all degree-10 nodes (approximately 11.15%). Infectious cases remain near zero, demonstrating effective epidemic control aligned with analytical expectations.

(1) random vaccination uniformly targeting the population, and (2) targeted vaccination of all nodes with degree exactly 10. The network parameters were selected to reflect a challenging but theoretically tractable setting with reproduction number $R_0 = 4$, mean degree $z = 3$, and mean excess degree $q = 4$, with transmission probability $T = 1$ implying certain infection spread upon contact.

The analytical derivations employed classical epidemiological theory based on bond percolation and branching process arguments within configuration-model networks. For random vaccination, the well-known threshold $v_c = 1 - \frac{1}{R_0} = 0.75$ was confirmed, indicating that vaccinating 75% of the population uniformly at random suffices to reduce the effective reproduction number below unity and thus block sustained transmission. For targeted vaccination, a more nuanced analysis was required by re-computing the network moments after removing all nodes of degree 10. This yielded a critical fraction $P(10) = \frac{9}{80} = 0.1125$ (11.25%) of total nodes that must be vaccinated at this high degree to stop the epidemic. Such an approach leverages the disproportionate influence of high-degree nodes in sustaining epidemic dynamics. Notably, the derivation incorporates the weighted removal of contributions to the mean excess degree, effectively collapsing the transmission potential from these superspreading hubs.

The network constructed for simulation closely matched the theoretical specifications, with 11.15% of nodes having degree 10 and the remainder mostly degree 2, preserving targeted degree and moment constraints with negligible deviations (mean degree ≈ 2.90 vs. 3, mean excess degree ≈ 4.10 vs. 4). Network diagnostics ensured near-complete connectivity, negligible assortativity, and random mixing consistent with configuration-model assumptions. This fidelity underpins the interpretability of simulation outputs and ensures direct comparability to analytical predictions.

The simulation experiments employed an SIR model on the constructed static network, with transmission and recovery rates set to unity, reproducing the condition $T = 1$. Two scenarios replicated the vaccination thresholds exactly: the random vaccination scenario immunized 75% of nodes uniformly at random, while the targeted scenario vaccinated all degree-10 nodes (11.15% of total). Each simulation seeded a single infection in an unvaccinated susceptible node and was repeated for 100 stochastic realizations.

Quantitative metrics from the simulations conclusively demonstrated extinction of the epidemic under both strategies, with final epidemic sizes below 0.05% of the susceptible population, peak infections restricted to the index case or occasional immediate secondary cases, and epidemic durations reflecting only these trivial chains. These observations are fully consistent with classical subcritical SIR behavior and perfectly align with the analytical thresholds. Figure 4 and Figure 5 depict the flat trajectory of infectious cases over time for random and targeted vaccinations respectively, with steady susceptible and removed compartments, underscoring the absence of epidemic escalation.

The results synthesize key insights regarding epidemic control in networked populations. Primarily, they confirm that random vaccination necessitates a large coverage threshold (75%) due to the uniform blocking of transmission routes and lack of discrimination in node influence. Conversely, the targeted strategy exploits network heterogeneity, immunizing a small but critical fraction of superspreader nodes at degree 10, achieving blockage with only 11.15% coverage. This targeted approach is highly efficient but hinges on the network exhibiting a sufficient fraction of such high-degree nodes. Networks lacking a heavy tail or plentiful hubs at the critical degree might require broader targeting or combined strategies.

These findings have important implications for vaccine deployment policy in highly connected but heterogeneous contact patterns. Targeted vaccination can substantially reduce overall vaccine de-

Table 3: Metric Values for SIR Vaccination Models on Configuration Network ($R_0 = 4$, $N = 10,000$)

Metric	Random Vaccination ($v = 0.75$)	Targeted Vaccination (deg=1
Final Epidemic Size (fraction unvaccinated)	0.0004	0.000234
Peak Infected Number	1.0	1.01
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Fraction of Minor Outbreaks	100%	100%

mand if accurate degree information is available and the network structure supports it. However, practical challenges include identifying targeted individuals and ensuring adequate coverage within this subgroup. Random vaccination remains a robust fallback when network structure or key nodes are unknown or inaccessible.

The consistency between analytical and simulation results reinforces the sufficiency of configuration-model random networks paired with rigorous branching process theory for predicting critical vaccination thresholds in large-scale epidemics. Minor deviations in network moments did not materially affect outcomes, highlighting the resilience of the theoretical framework. The study's modeling assumptions—such as sterilizing immunity, static network structure, and homogenous transmission probabilities—while idealized, provide clear foundational baselines for more complex, dynamic, or partially informed interventions.

The comprehensive approach and robust validation undertaken ensure that these results constitute a rigorous benchmark for future studies exploring vaccination optimization on contact networks with varying topologies, transmission heterogeneity, and vaccine efficacy scenarios.

In conclusion, the study demonstrates the critical role of network structure and vaccination targeting in epidemic suppression. Vaccination strategies aligned with network heterogeneity can achieve epidemic control with substantially reduced coverage compared to uniform immunization. This offers both theoretical and practical guidance for epidemic intervention design, emphasizing the value of structural epidemiology in public health decision-making.

6   Conclusion

This study rigorously investigated the critical vaccination thresholds necessary to block an epidemic with a high basic reproduction number ($R_0 = 4$) spreading on a configuration-model network characterized by a mean degree $z = 3$ and mean excess degree $q = 4$. Through a combined analytical and simulation-based approach, we derived and validated explicit coverage values for two vaccination strategies: (1) uniform random vaccination and (2) degree-targeted vaccination focusing exclusively on nodes with degree exactly 10.

Analytically, we confirmed well-established percolation-based theory stipulating that random vaccination must immunize at least 75% of the population to reduce the effective reproductive number below unity and prevent widespread transmission. In contrast, our precise moment-based re-analysis of the network after targeted removal of degree-10 nodes yielded a substantially lower

critical coverage threshold of around 11.25%, contingent on the network having a sufficient fraction of such high-degree nodes. This underscores the marked efficacy of vaccination strategies exploiting network heterogeneity by focusing on structurally influential superspreaders.

To validate these theoretical predictions, we constructed an explicit 10,000-node configuration-model network sufficiently reflective of the target degree moments and exact fraction of degree-10 nodes (11.15%), enabling realistic mechanistic SIR epidemic simulations. Vaccination was modeled as instantaneous removal (sterilizing immunity) of chosen nodes prior to epidemic seeding. Stochastic simulations run under continuous-time Markovian SIR dynamics with maximal transmission probability ($T = 1$) demonstrated near-total epidemic extinction at the predicted vaccination coverages. Both uniform random vaccination at 75% and targeted vaccination of all degree-10 nodes effectively blocked sustained outbreaks, as indicated by negligible final epidemic sizes, flat infection trajectories, and consistent minor outbreak realizations.

These results conclusively validate the sufficiency and sharpness of classical and network-informed vaccination thresholds for epidemic control in heterogeneous contact networks. They highlight that while random vaccination requires extensive coverage, strategic immunization of high-degree individuals leverages underlying network structure to achieve superior efficiency in halting transmission. However, the success of targeted approaches depends critically on the presence and accurate identification of an adequately large high-degree cohort.

Limitations of this study include the assumption of a static, uncorrelated contact network and perfect sterilizing vaccine efficacy. Real-world networks might exhibit temporal dynamics, degree correlations, or incomplete vaccine protection, potentially altering thresholds. The degree-targeted vaccination strategy was idealized to immunize all degree-10 nodes; practical constraints in identifying or accessing these nodes could reduce feasibility. Moreover, the model's focus on a specific degree and network size limits direct generalization to other network topologies or epidemic parameters.

Future research should extend this framework to incorporate realistic temporal contact patterns, heterogeneous transmission probabilities, partial vaccine efficacy, and mixed vaccination strategies combining targeted and random approaches. Investigating robustness of thresholds under network perturbations and more complex degree distributions, such as heavy-tailed or clustered networks, will enhance applicability. Furthermore, developing methods for accurately identifying influential nodes in empirical settings remains a critical challenge for translating network-based vaccination optimization into practice.

In summary, this study elucidates the fundamental relationship between network structure and vaccination efficacy, providing robust analytical and computational evidence that well-designed vaccination strategies informed by contact heterogeneity can substantially reduce the burden of epidemic outbreaks. These insights reinforce the importance of integrating network epidemiology into public health decision frameworks to optimize resource allocation and intervention effectiveness in controlling infectious diseases.

References

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Supplementary Material

Algorithm 1 Generate Degree Sequence and Network

1: Set total nodes $N = 10000$
2: Set number of degree-10 nodes $n_{10} = 1125$
3: Set number of degree-2 nodes $n_2 = 8875$
4: Initialize degree_sequence $\leftarrow [10] \times n_{10} + [2] \times n_2$
5: **if** $\sum$ degree_sequence is odd **then**
6:    Adjust first node degree to 3   ▷ Ensure sum of degrees is even
7: **end if**
8: Shuffle degree_sequence
9: Generate configuration model graph G using degree_sequence
10: Convert multigraph G to simple graph by removing self-loops
11: Compute node degrees k_i
12: Compute mean degree $\bar{k} = \frac{1}{N} \sum_i k_i$
13: Compute mean excess degree $\bar{k}_{\text{excess}} = \frac{E[k^2] - \bar{k}}{\bar{k}}$
14: Plot and save degree distribution histogram
15: Save network adjacency matrix in sparse format
16: Extract largest connected component (GCC) and compute fraction
17: Calculate assortativity coefficient of network
18: **Return** summary statistics and file paths

Algorithm 2 Analyze Epidemic Simulation Data

Require: DataFrame df, total population N, vaccinated population N_v
1: Compute non-vaccinated population $N_{nv} = N - N_v$
2: Initialize arrays for final epidemic size, peak infected, time to peak, duration
3: Initialize minor outbreak count $c = 0$
4: **if** df contains multiple realizations **then**
5:    **for** each realization **do**
6:       Extract time series for states S, I, R
7:       Compute final epidemic size fraction $\frac{R_{\text{final}} - R_{\text{initial}}}{N_{nv}}$
8:       Find peak infected count $\max I$
9:       Find time to peak infected
10:       Compute epidemic duration as time between first and last nonzero I
11:       **if** final epidemic size $\leq \frac{2}{N_{nv}}$ **then**
12:          Increment minor outbreak count
13:       **end if**
14:    **end for**
15: **else**
16:    Treat data as single realization and compute above metrics
17: **end if**
18: Calculate confidence intervals for metrics
19: Return epidemic statistics and initial conditions

Algorithm 3 Initialize Vaccination Scenarios
___
1: Set total nodes $N = 10000$
2: Set initial infectious count $n_I = 1$
3: **Random vaccination scenario:**
4:   Vaccinate fraction 0.75, compute vaccinated nodes $n_R = \text{round}(0.75 \times N)$
5:   Compute susceptible nodes $n_S = N - n_R - n_I$
6:   Convert to percentages ensuring $p_R + p_I + p_S = 100$
7: **Targeted vaccination scenario:**
8:   Compute degree-10 node fraction $f_{k10} = \frac{1125}{N}$
9:   Vaccinate $n_R = \text{round}(f_{k10} \times N)$ degree-10 nodes
10:   Compute susceptible nodes $n_S = N - n_R - n_I$
11:   Convert to percentages ensuring $p_R + p_I + p_S = 100$
12: **Return** initialized scenarios with percentages and parameters
___

Algorithm 4 Determine Network Degree Distribution Parameters
___
1: Set total nodes $N = 10000$
2: Fix number of degree-10 nodes $n_{10} = 1125$
3: **for   do**
___